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Original article

The number of leads with fragmented QRS is independently associated with cardiac death or hospitalization for heart failure in patients with prior myocardial infarction

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KEYWORDS
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Summary
Background: No information is currently available on the prognostic significance of the number of leads with fragmented QRS (fQRS). The objective of the study was to clarify the prognostic significance of the number of leads with fQRS in prior myocardial infarction (MI).
Methods and results: We retrospectively examined 170 patients with prior MI. The primary end point was cardiac death or hospitalization for heart failure. During a mean follow-up period of 6.4 ± 2.9 years, 37 patients developed the primary end point. Univariate Cox proportional hazards regression analyses showed that age, male gender, chronic kidney disease, anterior wall MI, number of leads with fQRS, left ventricular ejection fraction, loop diuretic use, and spironolactone use were significantly associated with the primary end point. A multivariate Cox proportional hazards regression analysis selected age (hazard ratio [HR] 1.09, 95% confidence interval [CI] 1.04—1.14, $p < 0.001$) and the number of leads with fQRS (HR 1.33, 95% CI 1.11—1.60, $p = 0.002$) as predictors of the primary end point. A receiver operating characteristic curve analysis showed that the presence of ≥3 leads with fQRS was most useful for distinguishing between patients with and without the primary end point. A Kaplan—Meier analysis showed a lower primary event-free rate in patients with ≥3 leads with fQRS than in those with <3 leads with fQRS.
Conclusions: The number of leads with fQRS, especially the presence of ≥3 leads with fQRS, is an independent predictor of cardiac death or hospitalization for heart failure in patients with prior MI.

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Introduction

Regional myocardial scars have been suggested to be associated with alterations in QRS morphology, leading to terminal conduction delay or fragmentation of the QRS complexes on the 12-lead electrocardiogram [1,2]. Das et al. [3] demonstrated that the presence of fragmented QRS (fQRS), defined as an additional R wave (R'), notching of the R wave, notching of the downstroke or upstroke of the S wave, or the presence of >1R' in ≥2 contiguous leads corresponding to a major coronary artery territory on the 12-lead electrocardiogram, is associated with myocardial scars on myocardial perfusion imaging in patients with coronary artery disease. Furthermore, the presence of fQRS has been shown to predict adverse outcomes in patients with coronary artery disease [4], acute coronary syndrome [5], prior myocardial infarction (MI) showing resolved Q waves [6], and ischemic or non-ischemic cardiomyopathy [7]. However, no information is currently available about the prognostic significance of the number of leads with fQRS. We hypothesized that the number of leads with fQRS would be associated with cardiac death or hospitalization for heart failure in patients with prior MI. The present study was conducted to test this hypothesis.

Methods

Patient population

Eligible patients with prior MI (>6 months) who had undergone diagnostic or follow-up coronary angiography together with left ventriculography at Oita University Hospital between January 2000 and December 2006 were enrolled in this retrospective study. The exclusion criteria were as follows: (1) critical stenotic lesions in the major coronary arteries requiring revascularization; (2) concomitant myocardial diseases or valvular heart diseases requiring cardiac surgery; (3) bundle branch block; and (4) life-threatening non-cardiac diseases. A prior MI was defined as follows: (1) a history of acute MI, which was identified by a rise in serum creatine kinase together with ≥1 of the following conditions: ischemic chest pain lasting ≥20 min; electrocardiographic changes indicative of new myocardial ischemia; development of abnormal Q waves; total or subtotal occlusion of the coronary artery on coronary angiograms; or cardiac imaging evidence of new loss of viable myocardium or new regional wall motion in the left ventricle; or (2) irrespective of a history of ischemic chest pain lasting ≥20 min, abnormal Q waves and imaging evidence of loss of viable myocardium in the left ventricle in the absence of a non-ischemic cause. The study protocol was approved by the ethics committee at our institution.

Data collection and primary end point

The following data at baseline were collected from medical records: age, gender, body mass index, presence or absence of current smoking, hypertension, diabetes mellitus, dyslipidemia, chronic kidney disease, prior percutaneous coronary intervention, prior coronary bypass surgery, and multivessel disease, left ventricular (LV) ejection fraction obtained from left ventriculography, and medications. Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, or treatment with antihypertensive drugs. Diabetes mellitus was defined as fasting plasma glucose ≥126 mg/dl, plasma glucose ≥200 mg/dl at 2 h after a 75 g glucose load, or treatment with hypoglycemic agents. Chronic kidney disease was defined as an estimated glomerular filtration rate <60 ml/min/1.73 m^2. The estimated glomerular filtration rate was calculated by the equation proposed by the Japanese Society of Nephrology [8]. Multivessel coronary disease was defined as the presence of luminal diameter stenosis >50% in ≥2 coronary arteries.

The primary end point was cardiac death or hospitalization for heart failure on or before December 31, 2010. Cardiac death included sudden death. Cardiac death or hospitalization for heart failure was documented from hospital medical records and information obtained by patients or patients' relatives using a direct or telephone interview.

Electrocardiography

A standard 12-lead electrocardiogram was recorded at a paper speed of 25 mm/s, an amplification of 10 mm = 1 mV, and a filter range from 0.1 to 150 Hz. The fQRS was defined as an R', notching of the R wave, notching of the downstroke or upstroke of the S wave, or the presence of >1R' [3]. An abnormal Q wave was defined as a wave of ≥20 ms in duration or a QS complex in leads V_{2-3} and a wave of ≥30 ms in duration and ≥1 mm in depth or a QS complex in other leads [9,10]. The presence or absence of abnormal Q waves and fQRS was determined by the consensus of 2 observers who were blinded to all of the patients' clinical and angiographic data. Inter- and intra-observer concordance rates for the number of leads with fQRS in each patient were 97.6% and 96.5%, respectively.

Statistical analysis

Data are expressed as mean ± SD or *n* (%). Comparisons of continuous variables were performed using the unpaired *t* test or the Mann–Whitney *U* test. Comparisons of categorical variables were performed using the chi-square test. Univariate and multivariate analyses using a Cox proportional hazards model were performed to determine the predictors of the primary end point. The following explanatory variables were used in the univariate analyses: age, male gender, body mass index, diabetes mellitus, current smoking, chronic kidney disease, anterior wall MI, multivessel coronary disease, LV ejection fraction, number of leads with abnormal Q waves, number of leads with fQRS, loop diuretic use, spironolactone use, renin–angiotensin system inhibitor (angiotensin-converting enzyme inhibitor or angiotensin receptor blocker) use, beta-blocker use, and statin use. Explanatory variables with a *p* value <0.1 on the univariate analyses were entered into a multivariate analysis. A receiver operating characteristic curve (ROC) was used to determine the best cut-off number of leads with fQRS for distinguishing between patients who developed the primary end point and those who did not. The best cut-off number

Table 1 Baseline characteristics.

Number of patients	170
Age (years)	68.6 ± 9.2
Male gender	136 (80.0)
Body mass index (kg/m^2)	23.7 ± 3.0
Hypertension	141 (82.9)
Dyslipidemia	141 (82.9)
Diabetes mellitus	75 (44.1)
Current smoking	22 (12.9)
Chronic kidney disease	82 (48.2)
Anterior wall MI	92 (54.1)
Multivessel coronary disease	101 (59.4)
Prior percutaneous coronary intervention	163 (95.9)
Balloon angioplasty	137 (80.6)
Stenting	93 (54.7)
Rotablation	13 (7.6)
Prior coronary artery bypass graft	20 (11.8)
Number of leads with abnormal Q waves	2.6 ± 1.9
Number of leads with fQRS	1.9 ± 2.0
LV ejection fraction (%)	53.9 ± 14.0
Medications	
Aspirin	123 (72.4)
Loop diuretic	38 (22.4)
Spironolactone	28 (16.5)
Calcium antagonist	79 (46.5)
Renin–angiotensin system inhibitor	94 (55.3)
Beta-blocker	57 (33.5)
Statin	121 (71.2)

Data are expressed as mean ± SD or *n* (%).
MI, myocardial infarction; fQRS, fragmented QRS; LV, left ventricular.

was defined as the point with the highest sum of sensitivity and specificity. The event-free rate was estimated using a Kaplan–Meier analysis, and the event-free curves were then compared using the log-rank test. A *p*-value <0.05 was considered to be statistically significant. The data analysis was carried out using the SPSS 12.0J software program for Windows (SPSS Inc., Tokyo, Japan).

Results

One hundred seventy patients (136 males and 34 females, mean age of 68.6 ± 9.2 years) were included in this study. Of 170 patients, 2 could not be followed up until December 31, 2010. Table 1 shows the patient characteristics at baseline. Fig. 1 shows the distribution of the number of leads with fQRS. A fQRS was observed in ≥1 lead(s) in 115 (67.6%) patients.

During a mean follow-up period of 6.4 ± 2.9 years, 37 patients developed the primary end point: cardiac death in 7 (sudden death in 4, fatal MI in 2, and ventricular fibrillation in 1) and hospitalization for heart failure in 30 (followed by death due to heart failure during hospitalization in 10). Patients with the primary end point had a larger number of leads with fQRS than those without (3.5 ± 2.2 vs 1.5 ± 1.7, $p < 0.001$). Table 2 shows the results of univariate and multivariate Cox proportional hazards regression analyses. Univariate Cox proportional hazards regression analyses showed that age, male gender, chronic kidney disease,

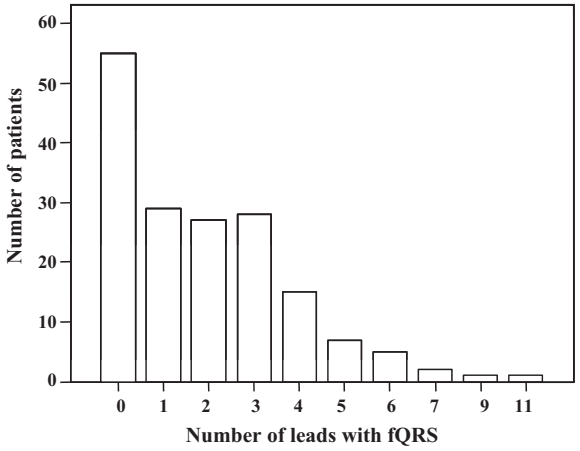

Figure 1 The distribution of the number of leads with fragmented QRS (fQRS).

anterior wall MI, LV ejection fraction, number of leads with fQRS, loop diuretic use, and spironolactone use were significantly associated with the primary end point. A multivariate Cox proportional hazards regression analysis showed that age (hazard ratio [HR] 1.09, 95% confidence interval [CI] 1.04–1.14, $p < 0.001$) and number of leads with fQRS (HR 1.33, 95% CI 1.11–1.60, $p = 0.002$) were independent predictors of the primary end point.

The best cut-off number of leads with fQRS to distinguish between patients who developed the primary end point and those who did not was 3 (Fig. 2). This cut-off number yielded a sensitivity of 70.3% and a specificity of 75.2% for distinguishing between the 2 groups of patients. The event-free rate was significantly lower in patients with ≥3 leads with fQRS than in those with <3 leads with fQRS ($p < 0.001$) (Fig. 3). The event-free rate was also significantly lower in the former than in the latter in both patients with anterior wall MI ($n = 92$) and those with non-anterior wall MI

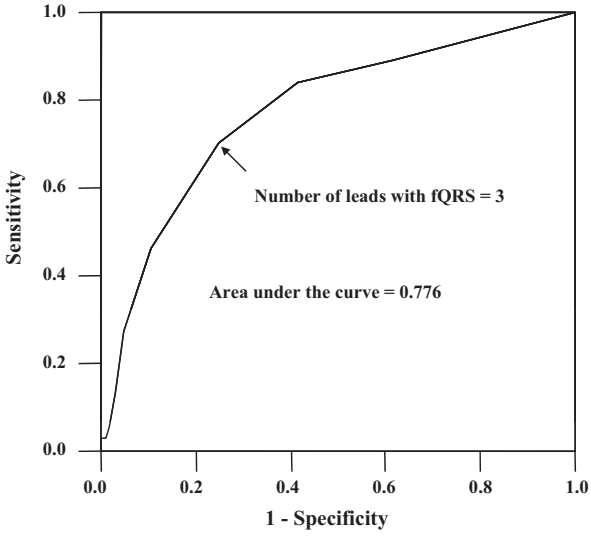

Figure 2 A receiver operating characteristic curve to determine the best cut-off number of leads with fragmented QRS (fQRS) for distinguishing between patients with and without the primary end point.

Table 2 Univariate and multivariate analyses using a Cox proportional hazards model to determine the predictors of the primary end point.

Variable	Univariate		Multivariate	
	HR (95% CI)	*p*-Value	HR (95% CI)	*p*-Value
Age (years)	1.10 (1.05—1.15)	<0.001	1.09 (1.04—1.14)	<0.001
Male gender	0.42 (0.21—0.82)	0.01	0.65 (0.30—1.40)	0.27
Body mass index (kg/m^2)	0.93 (0.84—1.03)	0.16		
Diabetes mellitus	1.43 (0.75—2.72)	0.28		
Current smoking	0.17 (0.02—1.21)	0.08	0.39 (0.05—3.13)	0.38
Chronic kidney disease	2.66 (1.31—5.38)	0.007	1.51 (0.67—3.36)	0.32
Anterior wall MI	2.73 (1.29—5.79)	0.009	1.28 (0.52—3.17)	0.60
Multivessel coronary disease	1.99 (0.96—4.11)	0.06	1.53 (0.69—3.40)	0.30
LV ejection fraction (%)	0.95 (0.93—0.97)	<0.001	1.00 (0.96—1.03)	0.74
Number of leads with abnormal Q waves	1.12 (0.97—1.29)	0.12		
Number of leads with fQRS	1.52 (1.33—1.75)	<0.001	1.33 (1.11—1.60)	0.002
Loop diuretic	6.03 (3.13—11.61)	<0.001	2.54 (0.87—7.44)	0.09
Spironolactone	3.89 (1.99—7.60)	<0.001	1.02 (0.38—2.72)	0.97
Renin—angiotensin system inhibitor	0.88 (0.46—1.69)	0.71		
Beta-blocker	1.27 (0.66—2.48)	0.48		
Statin	0.77 (0.39—1.54)	0.47		

HR, hazard ratio; CI, confidence interval; MI, myocardial infarction; fQRS, fragmented QRS; LV, left ventricular.

($n = 78$) (Fig. 4). Patients with ≥3 leads with fQRS ($n = 59$) had higher prevalences of anterior wall MI, prior coronary artery bypass graft, loop diuretic use, and spironolactone use, a lower prevalence of calcium antagonist use, greater numbers of leads with abnormal Q waves, and lower LV ejection fractions than those with <3 leads with fQRS ($n = 111$) (Table 3).

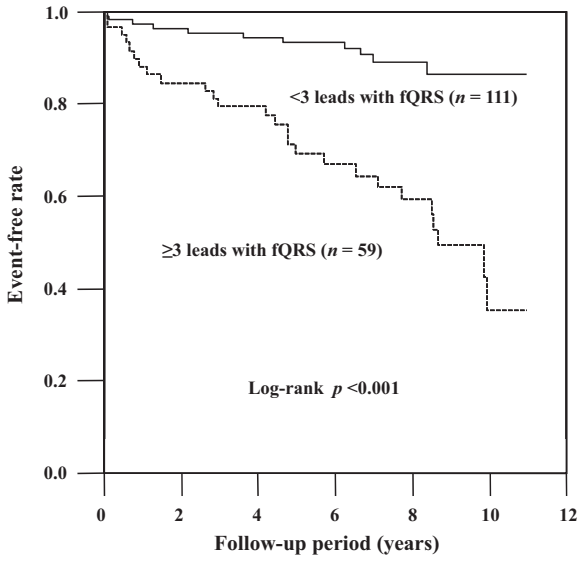

Figure 3 The Kaplan—Meier primary event-free curves for patients with ≥3 leads with fragmented QRS (fQRS) and those with <3 leads with fQRS.

Discussion

To the best of our knowledge, this is the first study that has examined the prognostic significance of the number of leads with fQRS in patients with prior MI.

The presence of fQRS in ≥2 contiguous leads corresponding to a major coronary artery territory on the 12-lead electrocardiogram has been shown to predict cardiac events in patients with coronary artery disease [4], all-cause mortality in those with acute coronary syndrome [5], cardiac events in those with prior MI showing resolved Q waves [6], and all-cause mortality and arrhythmic events in those with ischemic or non-ischemic cardiomyopathy [7]. It has been considered that fQRS results from the presence of significant myocardial necrosis, with islands of viable myocardial tissue interspersed in abundant fibrous tissue in coronary artery disease [11]. It would be expected that a larger number of fQRS may be associated with a larger size of myocardial scars [3,12], a more depressed LV systolic function [12], and intraventricular systolic dyssynchrony [13]. We thus hypothesized that a larger number of leads with fQRS would be associated with cardiac death or hospitalization for heart failure in patients with prior MI. In the present study, the number of leads with fQRS was significantly higher in MI patients who developed cardiac death or hospitalization for heart failure than in those who did not. A multivariate Cox proportional hazards regression analysis selected the number of leads with fQRS as one of the independent predictors for cardiac death or hospitalization for heart failure. These findings suggest that the number of leads with fQRS provides useful prognostic information for patients with prior MI. Furthermore, in the present study, the event-free rate was significantly lower in patients with ≥3 leads with fQRS

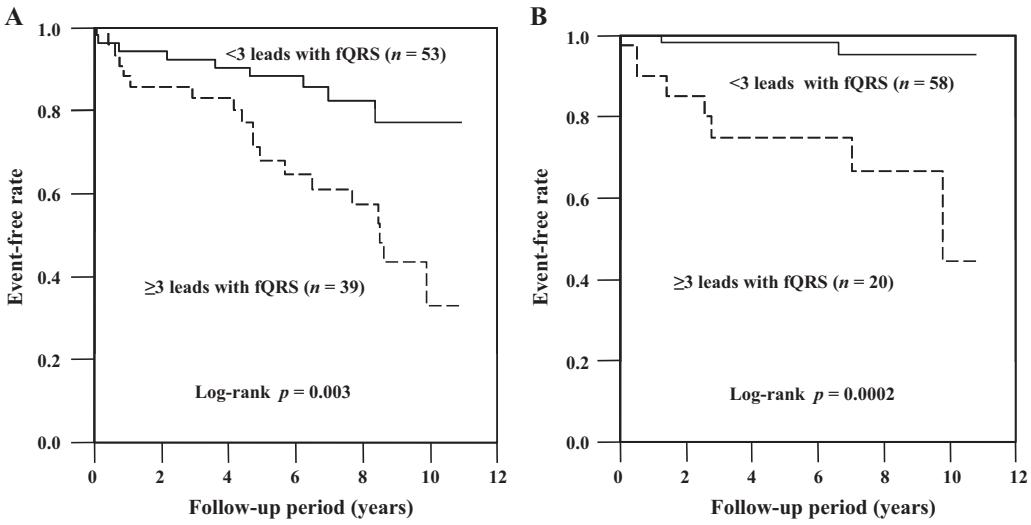

Figure 4 The Kaplan–Meier primary event-free curves for patients with ≥3 leads with fragmented QRS (fQRS) and those with <3 leads with fQRS in those with anterior wall myocardial infarction (MI) (A) and those with non-anterior wall MI (B).

than in those with <3 leads with fQRS in each group of all patients, those with anterior wall MI, or those with non-anterior wall MI. This cut-off number of leads with fQRS will need to be validated in prospective studies with a large sample size.

Because the myocardial scar can serve as an arrhythmogenic substrate in patients with prior MI, a larger number of leads with fQRS may indicate a high risk for sudden death due to life-threatening arrhythmia. Das et al. [7] demonstrated that the presence of fQRS in ≥2 contiguous leads was an independent predictor of arrhythmic events in patients with ischemic or non-ischemic cardiomyopathy who received an implantable cardioverter-defibrillator. In contrast, Cheema et al. [14] found no significant association between the presence of fQRS in ≥2 contiguous leads and arrhythmic mortality in ischemic or non-ischemic cardiomyopathy. In the present study, we could not analyze the association between the number of leads with fQRS and arrhythmic

Table 3 Comparison of baseline characteristics between patients with ≥3 leads with fQRS and those with <3 leads with fQRS.

Variable	≥3 leads with fQRS ($n = 59$)	<3 leads with fQRS ($n = 111$)	p-Value
Age (years)	69.7 ± 9.6	68.0 ± 9.0	0.23
Male gender	47 (79.7)	88 (79.3)	0.95
Body mass index (kg/m^2)	23.6 ± 3.0	23.8 ± 3.0	0.73
Hypertension	50 (84.7)	91 (82.0)	0.65
Dyslipidemia	49 (83.1)	92 (82.9)	0.98
Diabetes mellitus	31 (52.5)	44 (39.6)	0.11
Current smoking	4 (6.8)	18 (16.2)	0.10
Chronic kidney disease	34 (57.6)	48 (43.2)	0.07
Anterior wall MI	39 (66.1)	53 (47.7)	0.02
Multivessel coronary disease	38 (64.4)	63 (56.8)	0.33
Prior percutaneous coronary intervention	57 (96.6)	106 (95.5)	0.99
Prior coronary artery bypass graft	11 (18.6)	9 (8.1)	0.04
Number of leads with abnormal Q waves	3.3 ± 2.1	2.2 ± 1.8	0.001
LV ejection fraction (%)	44.9 ± 13.0	58.7 ± 12.1	<0.001
Medications			
Aspirin	43 (72.9)	80 (72.1)	0.91
Loop diuretic	26 (44.1)	12 (10.8)	<0.001
Spironolactone	18 (30.5)	10 (9.0)	<0.001
Calcium antagonist	21 (35.6)	58 (52.3)	0.04
Renin–angiotensin system inhibitor	36 (61.0)	58 (52.3)	0.27
Beta-blocker	25 (42.4)	32 (28.8)	0.08
Statin	39 (66.1)	82 (73.9)	0.29

Data are expressed as mean ± SD or n (%).
MI, myocardial infarction; fQRS, fragmented QRS; LV, left ventricular.

mortality because of the small number of patients with definite arrhythmic death ($n = 1$). Whether the number of leads with fQRS is associated with arrhythmic deaths in patients with prior MI will need to be investigated in prospective studies with a larger number of patients.

In the present study, the number of leads with abnormal Q waves was not associated with cardiac death or hospitalization for heart failure. This might be because an abnormal Q wave is an insensitive marker of myocardial scars [15—17]. A recent study reported that the number of leads with Q waves after primary percutaneous coronary intervention for ST-segment elevation acute MI was a strong predictor of mortality [18]. However, the definition of Q waves used in that study (an initial negative deflection $\geq$0.1 mV in a lead with ST-segment elevation $\geq$0.1 mV on the initial electrocardiogram) was different from the standard definition of abnormal Q waves [9], which was used in the present study.

Study limitations

The present study has a few limitations. First, this was a retrospective study that included a relatively small number of patients with prior MI. Therefore, the results of the present study will need to be validated in prospective studies including a large sample size. Second, the prevalence of beta-blocker use and renin—angiotensin inhibitor use at baseline was not high in the present study, although some patients started treatment with these agents during the follow-up period. Therefore, the results of the present study may not be applicable to patients with prior MI who undergo optimal pharmacological treatment. Third, the present study excluded patients with bundle branch block. Fragmented wide QRS was previously reported to be an independent predictor of mortality in patients with known or suspected coronary artery disease [19]. The prognostic significance of the number of leads with fragmented wide fQRS will need to be clarified in patients with prior MI. Finally, further studies with a large sample size are needed to determine whether the number of leads with fQRS is a more useful index for predicting cardiac death or hospitalization for heart failure in patients with prior MI than other electrocardiographic indices including Selvester score.

Conclusions

The present study is the first to show the number of leads with fQRS, especially the presence of $\geq$3 leads with fQRS, to be independently associated with cardiac death or hospitalization for heart failure in patients with prior MI.

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